suggested, this should not stop us from introducing Mn to gasoline because the balance for and against doing so is clearly in favor of approving Ethyl's application.

Best regards.

Sincerely yours,

Henry M. Wisniewski, M.D., Ph.D.
Director

CURRICULUM VITAE

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Education: M.D., Medical School, Gdansk (Danzig), Poland, 1955; Ph.D., Medical School, Warsaw, Poland, Experimental Pathology, 1960; Docent, Neuropathology, Medical School, Warsaw, Poland, 1965.

Honors:
Weil Award, American Association of Neuropathologists, 1969
Moore Award, American Association of Neuropathologists, 1972
Career Scientist, Health Research Council of the City of New York
Association for the Help of Retarded Children, New York City (AHRC) Chapter award, 1984
President, American Assoc. of Neuropathologists, 1984
Welfare League, Letchworth Village Chapter, AHRC, award 1985
Benevolent Society for Retarded Children, Staten Island Chapter, 1986
Fellow, American Association for the Advancement of Science, 1989

Experience:
Director, NYS Institute for Basic Research in Developmental Disabilities, 1976--present;
Professor of Pathology (Neuropathology), SUNY Health Science Center at Brooklyn, 1976--present;
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Research Assoc., Assistant, Associate and full Professor of Pathology (Neuropathology), Albert Einstein College of Medicine, 1966-1975;
Visiting Scientist, Laboratory of Neuropathology NINCDS, NIH (Dr. Klatzo), 1962-63;
Visiting Neuropathologist, Division of Neuropathology, University of Toronto, Canada (Dr. J. Olszewski) 1961-1962;
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Societies:
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British Immunological Association
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Other Activities:
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PUBLICATIONS

HENRYK M. WISNIEWSKI

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ATTACHMENT 2

MANGANESE : COMMENTS ON HUMAN HEALTH RISK

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For the general population, food usually constitutes the major source of manganese intake. The daily intake from food may vary over a wide range. In 1982, we have performed a duplicate meal study in Belgium and found values ranging from 0.6 to 8.8 mg manganese in 24-hr diets (median : 2.6). Drinking water generally contains less than 100 µg manganese/l (median value around 5 µg/l) but some mineralized water may contain higher concentrations. The median intake via drinking water is about 8 µg/day but can be as high as 2 mg/day from some water supplies. In many countries legislation requires that drinking water does not contain more than 50 µg manganese per liter. The adequate daily oral intake of manganese has been estimated at 2-3 mg but an additional amount of manganese (usually 1 mg po daily) is frequently prescribed during pregnancy in association with other oligoelements and various vitamins. Manganese absorption from the gastrointestinal tract is controlled by homeostatic mechanisms and varies from 1 to 5% (3% on the average). Individuals ingesting 3 mg manganese with food and drinking water will thus absorb on the average 90 µg manganese daily (range : 30 to 150 µg).

For non occupationally exposed subjects, manganese in ambient air represents a minor source of exposure. In rural and urban areas without significant manganese pollution, annual average levels of manganese in ambient air are mainly in the range of 0.005 - 0.07 µg/m³. With local industrial pollution, this level can rise to an annual average of 0.2 - 0.3 µg/m³ or even higher. Alveolar deposition of manganese may be estimated to be in the range of 0.07 - 0.5 µg/day as an average and 6-7 µg/day under high environmental exposure conditions. Even if the total amount deposited in the lungs is absorbed, the amount of manganese entering the circulation daily from the respiratory tract is much lower than that absorbed from the gastrointestinal tract.

Manganese is an essential element for mammals. It is incorporated in various enzymes and is a cofactor for a number of enzymatic reactions. Toxic effects of manganese have been mainly reported in workers chronically exposed to manganese dust and fume and more rarely among subjects living near ferromanganese plants (Elstad D. Norsk magazin for laegeridenskaben 3, 2527, 1939; Nogawa K. et al. Jpn J. Public Health 20, 315, 1973; Saric et al. International Conference on Heavy Metals in the Environment, vol. III, 27, 1975; Suzuki Y., Ind. Med. 12, 529, 1970) or drinking water contaminated by manganese (Kondakis et al. Arch. Environ. Health 44, 175, 1989). The two main target organs following chronic excessive exposure are the lungs (increased incidence of pneumonitis, bronchitis and chronic non specific lung disease) and the

central nervous system (neurobehavioral symptoms and neurological signs characteristic of an extrapyramidal syndrome).

The level of exposure to manganese which prevents the occurrence of central nervous system and lung disturbances has not yet been well defined. A WHO study group has concluded that there are indications that symptoms and signs which are not specific but may be connected with the early stage of the neurotoxic effects of manganese (manganism) can be found in a number of workers who have been exposed to manganese concentration of about 0.5 mg/m^3 of air; at approximately the same exposure levels, certain susceptible individuals may develop chronic manganese poisoning. Our recent studies on two cohorts of workers exposed to manganese oxides and salts support this conclusion. Adverse effects on the lungs of manganese exposed workers do not seem to appear at concentrations below 0.3-0.5 mg/m^3 of air. Studies performed on populations living near manganese emitting factories have suggested that an increased incidence of pneumonia and/or acute bronchitis may occur at lower levels of airbone manganese (references mentioned on page ?). It has been pointed out, however, that the increased incidence of pulmonary diseases found in these studies is not necessarily attributable to manganese itself. Other factors, including socioeconomic factors, which had not been considered may have influenced the results.

For occupational exposure, WHO (Geneva) has tentatively recommended a value of 0.3 mg of respirable manganese particles per m^3 of air as a

time-weighted average exposure. The preliminary results of an epidemiologic study among Belgian workers exposed to manganese dioxide in a dry alkaline-battery plant indicate that such exposure level may not necessarily prevents the occurrence of discrete neurofunctional changes in some subjects. This more recent study suggests that for occupational exposure it seems indicated to keep the airborne concentration (TWA) of respirable manganese particles below 150 µg/m³ (as manganese).

For the general environment, WHO (Air quality guidelines for Europe, WHO regional publications. European series; N° 23 - WHO Regional Office for Europe, Copenhagen 1987) has recommended an annual average of 1 µg manganese/m³ as a guideline value. This value should incorporate a sufficient margin of protection for the most sensitive population group.

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CURRICULUM VITAE

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Married

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Educational history

1962 : M.D. University of Louvain - Belgium

1965 : Master of Industrial Health - University of Louvain - Belgium

1966 : Master of Science in Hygiene (Toxicology)
Harvard University, Boston, USA

1968 : Doctor of Science in Hygiene (Toxicology)
Harvard University, Boston, USA

Positions

1962 - 1965 : Clinical assistant - Internal Medicine Department
University Hospital - Louvain - Belgium

1966 - 1968 : Research fellow : Harvard University (School of Public Health), Boston, U.S.A. (CRB Graduate fellow and U.S. Public Health Service fellow)

1968 - 1969 : Research assistant : Toxicology Research Unit
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Presently : - Professor of Occupational Health and Industrial Toxicology
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Scientific distinctions

. Delta Omega (U.S. Public Health Honorary degree)

. Prix Melsens (Académie Royale de Médecine de Belgique)

. Member Royal Medical Academy of Belgium

Member

- National organizations

. Council on Hygiene - Ministry of Health, Belgium

. Scientific Committee - Work Compensation Fund - Ministry of Social Affairs, Belgium

. Société Belge de Médecine Interne

. Cercle des Alumni de la Fondation Universitaire

- International organizations

. Council of Experts WHO

. Advisory Subgroup on Toxicology of the European Medical Research Council (1974-1988)

. CEC scientific committee on occupational exposure limits

. Toxicology section of CEC - Scientific Advisory Committee to examine the toxicity and ecotoxicity of chemical compounds

. Scientific Committee of "Centre d'Etudes et de Recherche des Charbonnages de France" from 1988

. European Society of Toxicology

. British Occupational Hygiene Society

. Society of Toxicology, USA

. American College of Occupational Medicine

. American Industrial Hygiene Association

. American Conference of Governmental Industrial Hygienists

. International Commission on Occupational Health

. Occupational Health in the Chemical Industry (Medichem)

. Participation at many international committees involved in the assessment of health risk due to environmental and/or industrial pollutants and in the guidance of research in this field

Member of the Editorial Board of

. Toxicology

. International Archives of Occupational and Environmental Health

. American Journal of Industrial Medicine (Foreign contributing editor)

. Toxicology Letters

. The Science of the Total Environment (from 1983 to 1987)

. Archives des Maladies Professionnelles

. Journal of Applied Toxicology

. Journal de Toxicologie Clinique et Expérimentale

. Archives of Environmental Health

. Toxicology and Applied Pharmacology (from 1976 to 1982)

. Cahiers des Notes Documentaires (INRS-France)

. Biological Monitoring

. Environmental Research

. Environmental Management and Health

Activities

Teaching industrial toxicology

Research in industrial toxicology

. study of the mechanism of action and the metabolism of industrial chemicals with the aim of developing methods for the early detection of adverse effects and assessing acceptable exposure levels

. epidemiological and experimental approaches

. main areas of research : metals, solvents, nephrotoxicity, biological monitoring of exposure.

Scientific publications :

Papers : 400 (English and French)

Books :

* Toxicologie Industrielle et Intoxications Professionnelles, Masson, Paris, 1990 (3rd edition) - First edition translated in Italian and in Finnish

* Industrial Chemical Exposure - Guidelines for Biological Monitoring. Biomedical Publications, Davis, California, 1983.

World Health Organization
Regional Office for Europe
Copenhagen

ATTACHMENT 3

Air quality guidelines for Europe

WHO Regional Publications, European Series No. 23

24

Manganese

General Description

Sources

Manganese (Mn) is an element widely distributed in the earth's crust. It is considered to be the twelfth most abundant element and the fifth most abundant metal. Manganese does not occur naturally in its native state. Oxides, carbonates and silicates are the most important among manganese-containing minerals. The most common manganese mineral is pyrolusite (MnO_2), usually mined in sedimentary deposits by opencast techniques. Manganese occurs in most iron ores. Its content in coal is in the range 6–100 μg/g; it is also present in crude oil, but at substantially lower concentrations *(1)*.

Manganese is mainly used in metallurgical processes, as a deoxidizing and desulfurizing additive and as an alloying constituent. It has other uses, e.g. in the production of dry-cell batteries, the production of manganese chemicals and in some other chemical processes, as well as in the manufacture of glass, in the leather and textile industries, and as a fertilizer. Organic carbonyl compounds are used as fuel-oil additives, smoke inhibitors, and as anti-knock additives in petrol *(2)*.

Crustal manganese enters the atmosphere by a number of natural and anthropogenic processes, which include the suspension of road dusts by vehicles, wind erosion and suspension of soils, particularly in agricultural and building activities and quarrying processes. The resulting mechanically generated aerosols consist primarily of coarse particles ($\geqq 2.5 \mu$m mass median aerodynamic diameter (MMAD)). The smelting of natural ores and the combustion of fossil fuels also result in the ejection of crustal manganese to the atmosphere in the form of fume or ash in the fine-particle range ($\leqq 2.5 \mu$m MMAD). Manganese is released to the atmosphere during the manufacture of ferroalloys and other industrial processes. Nearly one half of all industrial and combustive emissions of manganese are from ferroalloy manufacture and about one tenth from fossil-fuel combustion. Minor amounts are generated by other processes. The use of manganese fuel additives constitutes an additional source *(2)*.

As an element of low volatility, manganese tends to settle out near sources of pollution, but fine particles containing manganese can be distributed very widely. The most common forms of manganese compounds in the coarse particulates of crustal origin are oxides or hydroxides of oxidation state +2, +3, +4, and manganese carbonate. The manganese emitted by metallurgical processes consists of oxides. The manganese from combusted methylcyclopentadienyl manganese tricarbonyl (MMT), used in some countries as a fuel additive, is emitted primarily as Mn_3O_4. Minute amounts of organic manganese compounds may be present in ambient air under certain conditions. However, MMT is rapidly photodegraded to inorganic manganese in sunlight. The estimated half-time is 10–15 seconds *(2)*.

Manganese dioxide can react with sulfur dioxide or nitrogen dioxide to form manganous sulfate and dithionate, or manganese nitrate, respectively. It has been shown that aerosols of manganous sulfate can catalyse the oxidation of atmospheric sulfur dioxide to sulfur trioxide, thus promoting the formation of sulfuric acid *(2)*.

Occurrence in air

The natural level of manganese in air is low. Background concentrations of 0.05 to 5.4 ng/m³ over the Atlantic Ocean *(3)*, and 0.01 ng/m³ at the South Pole *(4)* have been reported. A concentration of 0.006 μg/m³ in air at a height of 2500 m and an annual average concentration of 0.027 μg/m³ at 823 m were reported *(5)*. The national air surveillance network of urban areas in the USA indicated an annual average manganese concentration of 0.033 μg/m³ in 1982 *(2)*. In two cities of the Federal Republic of Germany (Frankfurt am Main and Munich), annual mean concentrations of manganese ranged between 0.03 and 0.16 μg/m³ *(6)*, and in Belgium over the period 1972–1977 concentrations of manganese, expressed also as annual means, between 0.042 and 0.456 μg/m³ were reported *(7)*. The Environmental Agency Japan *(8)* reported an annual mean manganese concentration in the air of Japanese cities of about 0.02–0.80 μg/m³, with maximum 24-hour concentrations of 2–3 μg/m³.

From these and other data it can be concluded that annual average levels of manganese in ambient air in nonpolluted areas range from approximately 0.01 to 0.03 μg/m³, while in urban and rural areas without significant manganese pollution annual averages are mainly in the range of 0.01–0.07 μg/m³. With local pollution near foundries, this level can rise to an annual average of 0.2–0.3 μg/m³ and, in the presence of ferro- and silicomanganese industries, to over 0.5 μg/m³ *(9)*. In such places the average 24-hour concentrations may exceed 10 μg/m³.

About 80% of manganese emitted into the atmosphere is associated with particles with an MMAD of less than 5 μm, and about 50% with particles with an MMAD of less than 2 μm. More recent data, however, tend to indicate that less than 50% of the total measured manganese in ambient air is found in fine particles *(9)*.

Atmospheric particulate matter, including manganese, is transported by air currents until it is lost from the atmosphere by either dry or wet deposition. Manganese deposit in dustfall is more than twice that in rainfall *(2)*.

The highest values of manganese concentrations in the working environment have been reported from manganese mines, ore-processing plants, dry-cell battery plants, and ferromanganese plants. In mining operations, manganese concentrations up to 230 mg/m³ or even higher have sometimes been found. In dry-cell battery plants and ferromanganese plants the concentrations of manganese in air are lower. Values up to 5–8 mg/m³, but occasionally also higher — up to 20 mg/m³ or even more — have been reported *(10)*. An important point is that in ferromanganese plants, but also in dry-cell battery plants, the size distribution of manganese aerosols is such that small particles prevail absolutely, compared with mining operations, where a smaller proportion of respirable particles is usually encountered. There is also some evidence that aerosols formed by condensation may be more harmful than those formed by disintegration. Whether or not this is caused by differences in the distribution of particle size remains to be clarified *(10)*.

Routes of Exposure

Air

Because of the low solubility of manganese oxides, only inhaled manganese particles small enough to reach the alveoli (≤ 2.5 μm MMAD) are likely to enter the bloodstream. Alveolar deposition of manganese may be estimated to be in the range of 0.07–0.5 μg/day as an average and 6–7 μg/day under high-exposure conditions *(2)*.

Drinking-water

Concentrations of manganese in fresh water may vary from less than one to several thousand μg/litre *(11)*. Drinking-water generally contains less than 100 μg manganese per litre. In 100 of the largest cities in the USA, 97% of the surveyed public water supplies contained concentrations below 100 μg/litre.

Food

Manganese concentration in foodstuffs varies markedly, but on the whole food constitutes a major source of manganese intake for humans. The highest concentrations are found in some foods of plant origin, especially wheat and rice, with concentrations between 10 and 100 mg/kg. Polished rice and wheat flour contain less manganese, since most of it is in the bran. High concentrations of manganese have been found in tea leaves. Eggs, milk, fruits and meat generally contain less than 1 mg manganese per kg *(2)*. In a study performed in Canada *(12)* it was estimated that, of people's total manganese intake via food, 54% came from cereals and 14% from potatoes, whereas meat, fish and poultry provided only 2% of manganese intake. However, there is a difference in manganese concentrations for the same items in different countries and areas. Thus, daily manganese intake by adults from food may vary over a rather wide range (2–12 mg). The daily intake for children aged 3–5 years averages 1.4 mg/day and for children aged 9–13 years 2.18 mg/day *(13)*. The daily intake of manganese by bottle-fed and breast-fed infants is very low because of the low concentrations of manganese in both breast-milk and cow's milk *(14)*.

Relative significance of different routes of exposure

Total human exposure to manganese may be estimated from information on the levels in air, water and diet. The degree of uptake of manganese by inhalation is dependent upon particle size because of the low solubility of manganese oxides. Deposition of manganese in the alveoli can be calculated from the ambient concentration and the fraction present in fine particles. Alveolar deposition of manganese at current ambient levels may be estimated at 0.07 μg/day as an average and 6–7 μg/day under high-exposure conditions *(2)*. Thus, daily intake through inhalation generally constitutes less than 0.1% of the total daily intake by the general population and rarely exceeds 1%, even in heavily polluted areas *(1)*. Estimates of total thoracic deposition, including particles deposited in the tracheobronchial region, range from about 0.26 μg/day (average) to 10 μg/day (high) *(2)*.

Ingested manganese in diet is estimated to be 0.002–0.004 mg/kg body weight per day in infants and 0.06–0.08 mg/kg body weight per day in adults *(1)*. The daily intake of manganese from food by adults appears to be 2–9 mg. In Europe and the USA studies suggest a likely range of 2–5 mg, while in countries where grain and rice make up a major portion of the diet, the intake is more likely to be in the range of 5–9 mg. The consumption of tea may substantially add to the daily intake *(1)*. The median intake via drinking-water is about 0.008 mg/day, but can be as high as 2.0 mg/day for some water supplies. However, the average daily intake of manganese with water is unlikely to constitute more than 1–2% of the total intake of manganese. The ingestion of particles cleared from the respiratory tract probably constitutes no more than 0.01 mg/day under the highest ambient exposure conditions *(2)*.

Kinetics and Metabolism

There are no quantitative animal data on absorption rates for inhaled manganese compounds. Mena et al. *(15)* found that, in 17 humans exposed to a nebulized solution of manganese chloride and in 4 humans exposed to manganese oxide in a similar fashion, 40–70% of the deposited amount was recovered in the faeces. Both compounds were labelled with ^{54}Mn.

Manganese absorption is controlled by homeostatic mechanisms. The absorption rate will depend on the amount ingested and on tissue levels of manganese. In mice and rats, absorption of ingested manganese varies between 1 and 3.5%. It seems that manganese is absorbed equally well throughout the small intestine.

An increase in the iron content of milk decreases the whole-body retention of orally administered ^{54}Mn in rats by a factor of 10 *(16)*. Manganese interactions with other elements (cadmium, nickel, indium, rhodium, selenium) and ethanol at the level of gastrointestinal absorption were also observed.

Mena et al. *(15)* found that 11 normal individuals absorbed an average of 3% of a dose of 200 μg manganese chloride labelled with ^{54}Mn. Another human study *(17)* indicates that manganese absorption takes place by diffusion in iron overload states and by active transportation in the duodenum and jejunum in iron deficiency states.

Absorbed manganese is rapidly eliminated from the blood and at first concentrates mainly in the liver. Excess metal may be distributed to other tissues. Concentrations of manganese are characteristic of the individual tissues, and almost independent of the species. Manganese preferentially accumulates in tissues rich in mitochondria. It penetrates the placental barrier in all species and is secreted in milk *(2)*.

The highest concentrations of manganese in man have been found in the liver, kidneys, endocrine glands, and in the small and large intestines. The total body-burden for manganese has been estimated at 8–10 mg; about 35% of this amount is found in muscle tissue and 20% in the liver *(18)*.

In blood, manganese is bound to proteins. In the trivalent state it can bind to transferrin and in the divalent state to an α-macroglobulin. The organic compound MMT is rapidly metabolized. The distribution in general is similar to that seen after exposure to inorganic manganese *(1)*.

The apparent absence of cases of chronic oral manganese toxicity could be attributed to the extremely efficient homeostatic mechanism, well documented in animal and human studies, which prevents the accumulation of manganese in the body and maintains systemic plasma manganese concentrations at constant levels *(2,19)*. Manganese is excreted primarily via biliary clearance *(2)*. However, it is possible that the capacity of the liver to excrete higher amounts of inhaled or injected manganese is limited, resulting in an increase in the tissue retention of manganese and, consequently, in the manifestation of toxic effects *(19,20)*. In excessive exposure other gastrointestinal routes may participate. Urinary excretion is small; in humans it is less than 1 µg/day. This means that only about 0.01% of the body-burden is excreted daily via that route. Manganese is also excreted via sweat, hair, placenta and milk.

The biological half-time of manganese depends on the body-burden of manganese. Experiments have shown that manganese elimination from the brain is slower than that from the whole body. In humans without occupational exposure to manganese the half-time is around 35 days *(21)*.

Health Effects

Manganese is an essential element. It is a constituent of several enzymes and can also activate many enzymes. Manganese deficiency was described only once in a man given a synthetic diet in which manganese had been omitted by mistake. Among the symptoms and signs were dermatitis, pigment changes of hair, retarded hair growth and hypocholesterolaemia *(1)*. Excessive exposure to manganese has been shown to cause toxic effects in animals and humans. As manganese is regarded as a metal with a relatively low toxicity, acute poisoning by manganese in humans is very rare *(2)*.

Effects on experimental animals

A large number of studies on laboratory animals were performed in order to explain the mechanism underlying the neurotoxicity of manganese seen in exposed workers *(2)*. Although animal experiments show that in chronic

manganese intoxication the central dopaminergic system is disturbed, the mechanism of manganese neurotoxicity has not been elucidated *(2,22)*.

Recent studies indicate that age may play a role in the extrapyramidal disturbances seen in human manganism, suggesting that the aging brain shows differing susceptibility vis-à-vis the toxic effects of manganese *(2,23)*. However, some new studies raise the intriguing possibility that lifetime treatment with manganese has some beneficial effects on the aging brain *(23)*. An accurate dose–response relationship for inhalation exposure and neurotoxicity is unobtainable at present from the available animal studies *(2)*.

Inhalation studies of the pulmonary effects show the occurrence of acute respiratory effects (oedema and leukocyte infiltration) when the level of exposure exceeds 20 mg MnO_2 per m^3 *(2)*. Mice and monkeys exposed to MnO_2 by inhalation showed pathological effects after chronic (10 months) exposure to 0.7 mg/m^3 *(24)*. Studies in which animals were exposed for a long period of time (66 and 40 weeks) to about 0.1 mg MnO per m^3 as Mn_3O_4 particles or aerosols of respirable particle size showed no respiratory effects, but the studies had several methodological deficiencies which reduce confidence in the negative results *(25,26)*.

It is plausible that exposure to manganese may increase susceptibility to pulmonary infections by disturbing the normal mechanism of lung clearance. However, it seems that a primary inflammatory reaction can occur in the lung after exposure to MnO_2, if the concentration is high enough, without the presence of pathogenic bacteria *(27)*. Studies on the influence of manganese on susceptibility to bacterial infections showed increased morbidity and mortality rates in animals infected before, during or after exposure to manganese dioxide *(28,29)*.

Effects on humans

The neurological disorder known as manganism may result from occupational exposure to manganese dusts and fumes. Symptoms and signs of manganism have often been compared with Parkinson's disease, but certain differences should be noted. Patients with Parkinson's disease show pronounced disturbances of motor behaviour, which include tremor observed at rest rather than during intentional motor activity as in manganism. Fully developed manganism causes severe rigidity, with the extremities showing the "cog-wheel" phenomenon *(2,30)*. Manganism usually appears after a prolonged exposure of two or more years, but it may result from exposure to high concentrations of manganese for only a few months. The full clinical picture of chronic manganese poisoning has been reported mainly in manganese miners, but also in other occupations where exposure to manganese is high *(2,30)*. The disease has been found less frequently at exposure levels below 5 mg manganese per m^3. However, there are reports of signs and symptoms which may be connected with subclinical or early clinical stages of chronic manganese poisoning in workers exposed to 0.3–5 mg manganese per m^3. In connection with the toxic effects of manganese, a marked individual susceptibility has been observed *(10,30)*. The data available for identifying effects below 1 mg manganese particles per m^3 are equivocal

and inadequate. Furthermore, no good biological indicators of manganese exposure are available at present.

The toxic effects of manganese on the pulmonary system vary in type and severity. There are reports of humans developing pneumonia in occupational but also in ambient exposure to manganese. Increased incidence of pneumonia was observed in workers, mainly miners, exposed to manganese at levels higher than 5 mg/m³ *(2)*. In a more recent study *(31)*, an increased incidence of pneumonia and bronchitis was found in workers exposed to manganese concentrations of 0.4–16 mg/m³ in a factory producing manganese alloys.

Elstad *(32)* noted an 8-fold increase in mortality from pneumonia and a 4-fold increase in pneumonia morbidity in the general population living near a ferromanganese plant in Norway. The air concentrations of manganese were measured only once and were reported to be 46 μg/m³ at a distance of 3 km from the plant. It was also reported that the incidence of pneumonia followed the rate of production of manganese alloys. Analyses of lung tissues from 11 persons who died from pneumonia showed manganese concentrations of 0.35–1.63 μg/kg wet weight. A higher prevalence of nose and throat symptoms, and lower values of lung function tests compared with controls, were observed in children attending a school situated near a ferromanganese plant *(33)*, where the average manganese exposure was about 7 μg/m³ (range 3–11 μg/m³). The study involved several hundred children, had a participation rate of over 97% and documented monitored levels of settled manganese dust for several years. Effects of still lower levels of airborne manganese (about 1 μg/m³) have been claimed to occur in a population living near a manganese alloy plant, with an increased incidence of acute bronchitis over an observation period of 4 years. The incidence of pneumonia did not seem to exceed the expected values *(34)*. Although sulfur dioxide concentrations were measured (annual means: 10–30 μg/m³), other factors, including the socioeconomic factor, which had not been considered, may have influenced the results. Chronic bronchitis has been reported to be more prevalent in workers exposed to manganese if they are smokers *(35)*.

A number of effects of manganese in other organs and systems have been claimed on the basis of results obtained from animal experiments and from epidemiological and clinical studies. These include a decrease in systolic blood pressure values, an increased rate of spontaneous abortions, changes in erythropoiesis and granulocyte formation, disturbed excretion of 17-ketosteroids, and changes in the activity of some enzymes *(2)*. Reports of impotence in a number of patients with chronic manganese poisoning are also common. However, these effects have not been observed consistently. In some cases the implications of the results of animal experiments for human health are uncertain and therefore they cannot be regarded as relevant in the assessment of the potential health risk of ambient exposure to manganese.

Evaluation of Human Health Risks

Exposure

In urban and rural areas without significant manganese pollution, annual averages are mainly in the range of 0.01–0.07 μg/m³; near foundries the level

can rise to an annual average of 0.2–0.3 $\mu g/m^3$ and, where ferro- and silicomanganese industries are present, to more than 0.5 $\mu g/m^3$ with individual 24-hour concentrations exceeding 10 $\mu g/m^3$.

Health risk evaluation

Manganese is both an essential and, at higher levels, a toxic element. In assessing the health impact of manganese exposure, the effect on the CNS and the lungs should be regarded as the most significant. The neurological disorder known as manganism has been reported in the context of occupational exposure to manganese, seldom at levels below 5 mg/m^3. Studies related to signs and symptoms which may be connected with a subclinical or early stage of manganese poisoning in workers exposed to concentrations below 1 mg/m^3 are equivocal or negative.

Respiratory symptoms seem to occur at lower levels of exposure to manganese, e.g. below 1 mg/m^3, than do neurological symptoms and signs. Therefore, respiratory effects may be considered to be critical in ambient exposure to manganese. Schoolchildren exposed to about 7 μg manganese per m^3 (range 3–11 $\mu g/m^3$) emitted from a ferromanganese plant had an increased prevalence of respiratory symptoms. This level of 7 $\mu g/m^3$ may therefore be considered the lowest-observed-effect level *(33)*. This conclusion is supported by a report of increased incidence of acute bronchitis at levels of about 1 $\mu g/m^3$ in a population living near a manganese alloy plant *(34)*.

The available evidence indicates that the current manganese levels generally found in industrialized countries are not in the concentration range associated with potentially harmful effects.

Guidelines

Available data from epidemiological studies suggest that the lowest-observed-adverse-effect concentration of manganese is about 7 $\mu g/m^3$. It is assumed that below 1 $\mu g/m^3$ (annual average), adverse health effects of environmental exposure to manganese are not likely to occur and therefore an annual average of 1 $\mu g/m^3$ is recommended as a guideline value. This value incorporates a sufficient margin of protection for the most sensitive population group. As the critical effect is one of respiratory irritancy it is desirable to have a short-term guideline value, but the present data base does not allow such estimations.

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ATTACHMENT 4

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COMMENTS ON THE WAIVER APPLICATION BY ETHYL CORPORATION FOR METHYLCYCLOPENTADIENYL MANGANESE TRICARBONATE (MMT) AS A GASOLINE ADDITIVE.

These comments are submitted at the request of the Ethyl Corporation in support of its application for a waiver to permit the use of MMT as an additive to gasoline. I am an independent consultant based in Lafayette, California. In 1984 I wrote a comprehensive review of the scientific literature entitled "The Health Implications of Increased Manganese in the Environment Resulting from the Combustion of Fuel Additives. A Reiview of the Literature". It was published in the Journal of Toxicology and Environmental Health(Vol.14, pp 23-46).

The key questions considered in this review were:

"1. How would the increments of manganese intake predicted from MMT use relate quantitatively to normal background levels and to levels known to be toxic?

2. Are there differences in the absorption, distribution, and excretion of inhaled manganese, as contrasted with ingested manganese, which would make small increases in airborne manganese unusually hazardous?

3. Would individuals with iron-deficiency anemias be unusually susceptible because of increased absorption of Mn?

4. Are infants hypersusceptible, because of increased intestinal absorption and poorly developed blood-brain barriers to metals?

5. Are there effects, other than those on the central nervous system associated with high concentrations of Mn, that deserve consideration? These include acute respiratory disease, interference with hematopoiesis, reproductive problems, mutagenicity, and carcinogenicity.

6. From consideration of the above factors, is the use of MMT as a fuel additive acceptable in terms of the public's health? "

After reviewing all available environmental, experimental and epidemiologic evidence I reached the following conclusions:

1. The increments in manganese intake in humans resulting from the use of MMT would be within the physiologic range and far below those known to be toxic.

2. While there are differences in the absorption, distrib-

ution, and excretion of manganese that is inhaled as contrasted wuth manganese that is ingested, the differendes are relatively small. Animals that have inhaled manganese derived from the combustion of MMT in concentrations greatly in excess of any that would result from MMT's use as a gasoline additive have not shown toxic effects.

3. There is moderately increased absorption of Mn associated with iron deficiency anemias, but this related to ingested Mn. Any increments in ingested Mn from MMT use would be within the variations that normally occur from differing dietary intakes.

4. Very young experimental animals have increased intestinal absorption of Mn and poorly developed blood-brain barriers to metals. While this suggests that they might be hypersusceptible to central nervous system effects from manganese, the increments of Mn fromMMT use would lie within a range to which they are already being exposed and far below concentrations where such hypersusceptibility would be operative.

5. There is also no evidence to support any discernible impact of minute increments of Mn from MMT on the respiratory tract, the cardiovascular system, hematopoiesis, or reproduction. Neither should mutagenic or carcinogenic effects be anticipated, in view of the fact that total Mn intakes would remain in the physiologic range essential to health.

6. In spite of the fact that there are gaps in our knowledge of the metabolism of manganese and its functions and effects in biologic systems, these are more than balanced by experimental studies with high concentrations of Mn derived from the combustion of MMT. The minute increments of Mn that would result from the use of MMT as a gasoline additive should not have any impact on the public's health "

As of July 1990 I am not aware of any new evidence to alter these conclusions; if anything they have been strengthened. A review by Abbottt of the Australian Department of Health(1987) and another by the Health EffectsInstitute (1988) have been in essential agreement.

Some new studieds made available in the course of the recent hearing were reassuring. Roels et al (1987a, 1987b) reported epidemiologic surveys of Belgian workers exposed to inorganic Mn. Using very sensitive tests for neurotoxicity and respiratory effects, they reported only marginal changes at time-weighted averages of about 1,000 ug/m^3 over long periods of time.

The contributions by the Environmental Defense Fund(Dr. Ellen Silbergeld), Dr. Herbert L. Needleman, and Dr. David Hall added no evidence to support their positions against the waiver. The first two were largely devoted to criticism of Ethyl Corporation obviously related to the witnesses' long-standing concerns about lead.

The analogies between lead and manganese drawn by Drs. Silbergeld and Needleman are not scientifically valid or relevant to the current issue. They disregard the fact that manganese is essential to human health (acknowledged by Dr. Silbergeld on page 19), while lead is not. They ignore the fact that the range between lead exposures causing unquestionable toxicity and those where effects are minuscule is of the

order of 10:1 or less. On the other hand, for manganese conclusive neurotoxicity in occupationally-exposed workers are associated with exposures thousands of times greater than the concentrations expected to result from the use of MMT as a gasoline additive. The latter are within the range of background intake.

Some specific points raised by the witnesses deserve brief discussion. Dr. Silbergeld on page 3 criticized omission from Ethyl Corporation's presentation of a report from the California Air Resources Board(1989), saying that it "demonstrates that increases in airborne and deposited manganese are related to additions of manganese to gasoline already permitted in California". She did not point out that the report provided very weak evidence, limited to a few sites in Southern California to support the above. The authors concluded that "vehicular emissions of manganese may account for a significant part of the total at urban sites in Southern California ". Noteworthy in the report was the fact that nearly all the measured Mn levels were extremely low, the mean concentrations reported for the Central Valley, the San Francisco Bay Area, and Southern California being 30, 15, and 30 nanograms per cubic meter respectively. In nearly all sites the fraction attributed to vehicular emissions was much less than that from dust arising from the earth's crust.

Dr. Needleman's dramatic calculation of the total amount of manganese that would be contributed to the nation's air from the sale of 100 billion gallons of gasoline containing 31.2 mg of MMT per gallon was not particularly alarming The figure he arrived at was 30,000 kg. When much of the soil of the United States contains 1000 parts per million of manganese, 33 additional tons does not sound like very much.

The NIEHS contribution summarized toxicologic studies of MMT, the danger of which is unquestioned and which must be tightly controlled. However this toxicity, prior to addition to gasoline, is not at issue. Dr. Hall's comments were clearly of interest but in several areas did not consider evidence that is already in the record. His statement that epidemiolgoic studies on Mn have been primarily on humans exposed to large amounts of Mn-bearing dust/fumes (as in miners, metel workers, or grinders) is of course true . As he knows these have shown severe effects in workers exposed to 5,000 ug/m^3 of Mn or more. In the range 1000 ug/m^3 and up effects have been minor or equivocal. There are of course no studies in exposure ranges of 100 or 10 or 1 ug/m^3. How would anyone design an epidemiologic study in exposure levels comparable to our normal background?

A fundamental flaw in all of the arguments raised against the waiver has been a disregard for the importance of dose . Nearly anything is toxic in high enough concent rations. No one disuputes the fact that premature infants have been made totally blinded by postnatal exposure to 100% oxygen. Children can be killed by sodium chloride in amounts that seem surprisingly small.. Neurotixic effects from extremely high doses of Mn should not lead to the erroneous assumption that doses within the range normal background levels are hazardous. Even if some was in the tetroxide form and this were definitely more hazardous, we would still be dealing with extraordinarily low doses.

It is unfortunate that the opponents of the waiver have have paid so little attention to probable environmental benefits from the additive. The evidence for these seems far more convincing than that for hypothetical and improbable health risks from Mn.

I sincerely hope that the EPA considers the scientific evidence carefully in reaching its decision on the waiver.

Respectfully,

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1952-1957	Chief, Occupational Health Field Headquarters, Division of Occupational Health, PHS, Cincinnati, Ohio
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Fellow, Royal Society of Medicine (London)
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Member, Panel on Clinical Testing of Antimalarials, Board of Coordination of Malarial Studies, 1944-1946
Member, Threshold Limits Committee of the American Conference of Governmental Industrial Hygienists, 1956-1961
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Member, Task Force on Chronic Bronchitis and Emphysema (USPHS and National Tuberculosis Association), 1966-1967
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Member, Committee on Public Information in the Prevention of Occupational Cancer, National Academy of Sciences, 1975-1977
Member, Subcommittee on Occupational Health, Committee on Military Environmental Research, National Research Council, 1979-1981

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APPENDiX 8

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DETERMINATION OF METHYLCYCLOPENTADIENYLMANGANESE-TRICARBONYL BY GAS CHROMATOGRAPHY—ATOMIC ABSORPTION SPECTROMETRY AT ng m^{-3} LEVELS IN AIR SAMPLES

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SUMMARY

A procedure is given for the determination of methylcyclopentadienylmanganese tricarbonyl (MMT) at ng m^{-3} concentrations in air. The method involves trapping of MMT in a small segment of gas chromatographic column and then determination by gas chromatography with an electrothermal atomic absorption detector. The detection limit of the procedure is 0.05 ng m^{-3}. Air samples from an underground car-park (when MMT was detected) were found to contain between 0.1 and 0.3 ng m^{-3} MMT. MMT was not detected in any of the street air samples taken.

Methylcyclopentadienylmanganesetricarbonyl (MMT) is added to many of the so-called "unleaded" gasolines sold in North America, as an antiknock compound. Levels of MMT in these gasolines average about 0.16 g gal^{-1}. MMT was discovered and patented by Ethyl Corporation and there is little information in the scientific literature concerning this compound. The current widespread distribution of MMT as a gasoline additive makes it essential that a method be developed for its determination in environmental samples. Most procedures for the determination of MMT are indirect relating it to the total manganese found in a sample. Turkel'traub et al. [1] described a gas chromatographic procedure for the related compound cyclopentadienylmanganesetricarbonyl (CMT). Only one procedure, that of Uden et al. [2], has been reported for the direct determination of MMT. This latter procedure is for the relatively high levels of MMT found in gasoline. In the following paragraphs, a procedure is outlined for the gas chromatographic/atomic absorption determination of MMT directly at ng m^{-3} levels in air.

The techniques of analytical atomic spectrometry can be used to advantage in the study of metal compounds, as metal specific detectors for chromatography. Uden et al. [2] used d.c. argon plasma emission spectrometry as the detector in their work. The detection limit obtainable by d.c. argon plasma emission for manganese is poorer than for electrothermal atomic absorption. Thus this latter detector was chosen in the current work because of the very low levels of MMT expected in environmental samples.

EXPERIMENTAL

Apparatus and reagents

A Perkin-Elmer 603 atomic absorption spectrometer was used with a Perkin-Elmer HGA 2100 furnace. A deuterium arc background corrector was employed. A Pye gas chromatograph (Series 104) was interfaced to the graphite furnace using a tantalum connector as previously described [3]. A glass chromatographic column (2.3 m long, 6 mm o.d.) was packed with 3% OV-1 on high-performance Chromosorb W (80/100 mesh). The gas from the chromatograph was transferred to the furnace through teflon-lined aluminium tubing.

The gas chromatographic set up is illustrated in Fig. 1. (A) connects to a nitrogen cylinder. (B) is the sample oven containing the sample trap and 4-way valve. (C) is the gas chromatograph connected through a standard injection valve to the column. (D) is the graphite furnace. The operating conditions for the gas chromatograph and the graphite furnace were as shown in Table 1.

Samples of air were collected in teflon-lined aluminium U-tubes (30 cm long, 3 mm o.d.) packed with 3% OV-1 on Chromosorb W (80/100 mesh). These tubes were placed in a water—ice cooling bath. Air entered through an air filter previously described [3] and was pumped through the U-tube trap at about 70 ml min^{-1} using a vacuum pump. The length of sampling time and the average flow rate, checked frequently during sampling, were used to compute sample volume.

MMT (Alfa Division, Ventron Corporation, Danvers, Massachusetts) was diluted in isooctane to produce the working standards. These standards were prepared fresh daily.

Procedures

Air samples were collected for various periods of up to 100 h and were retained in a freezer until run.

The gas chromatographic system is assembled as shown in Fig. 1. All transfer lines between the sample oven (B) and the gas chromatograph (C) and thence to the graphite furnace (D), must be wrapped in 36-W heating tape and held at 150°C throughout the procedure.

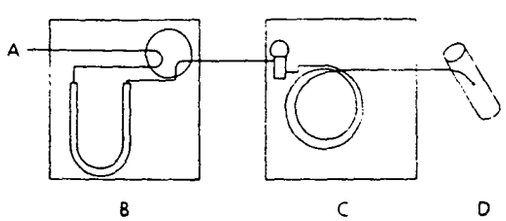

Fig. 1. Gas chromatograph system.

TABLE 1

Instrumental operating conditions

Gas chromatography		Atomic absorption spectrometry	
Carrier gas flow rate	80 ml min^{-1}	Ash 300°C	120 s (dry cycle)
Oven temperature program	115°C isothermal	Atomize 1800°C	240 s (char cycle)
Injection port temperature	150°C	Internal gas flow	0
Outlet and transfer tube temperature	150°C	External gas flow (N_2)	60 ml min^{-1}
		Wavelength	279.5 nm
		Slit 4	(0.7 nm)
		Background correction mode, Scale expansion (x 5)	

With the 4-way valve in the sample oven in the bypass position, the sample trap is placed in the cool sample oven and connected with Swagelok fittings. The nitrogen carrier gas flow rate is adjusted to 100 ml min^{-1} (which reduces to 80 ml min^{-1} when the sample trap is switched on). The sample oven is heated at 150°C for 10 min (no flow through the sample tube). The 4-way valve is then turned to start the flow through the sample tube, and the atomic absorption furnace program is initiated immediately. The gas chromatograph is run isothermally at 115°C. Gas flow is continued until the MMT peak has been recorded.

Calibration is done by injecting an appropriate μl standard of MMT directly into the cooled, spent, sample tube in the sample oven. The above procedure is then followed exactly as for the sample.

RESULTS AND DISCUSSION

Optimization of the procedure

Collection of MMT. Because of the very low levels of MMT expected in air and other environmental samples, it was necessary to employ a method of preconcentration. Two possibilities exist: (1) the MMT can be trapped in a solvent contained in a bubbler; or (2) the MMT can be trapped on an adsorber held at very low temperature. Both of these strategies were tried.

A micro bubbler containing hexane was cooled in an ice bath. After collection of the sample the hexane volume was reduced to 0.4 ml. A 10-μl aliquot was injected into the gas chromatograph. The detection limit using this approach was about 0.02 $\mu g\ m^{-3}$ of air for an 8-h sampling period. This was shown to be too poor for the present purposes.

The second trapping strategy is that outlined in the proposed procedure above. MMT is collected on a teflon-lined U-tube containing packing material similar to that used in the gas chromatographic columns. It was necessary to build a sample oven which contained a 4-way valve (which allows by-passing of the sample U-tube) so that any lines or other components carrying MMT

could be heated. This is because MMT has a relatively high boiling point and tends to condense out on cool transfer lines.

Decomposition of MMT in transfer lines. In a similar study of tetraalkyllead compounds, the problem of decomposition of these compounds in the metal transfer lines was noted. Teflon-lined tubing prevented this problem and was used as a precaution in this study as well.

Calibration. Ideally it would be desirable to inject standard solutions through the injection valve for calibration and avoid collection on the sample trap. However, the chromatographic peaks obtained by direct injection and those obtained using the sample trap had slightly different retention times and were differently shaped. Thus, it was necessary to inject the calibration standard onto the spent sample U-tube and follow the procedure exactly as for the samples. Peak area rather than peak height was used in the calculations.

Comparison of atomizers. Interfacing of gas chromatography and atomic absorption occurs through the atomizer. When best atomic absorption sensitivity is required, a commercial electrothermal atomizer (furnace or rod) is usually chosen. These devices are relatively expensive and are not available in all atomic absorption laboratories to be tied up for this purpose. Thus it was of interest to determine the relative detection limits obtainable for inexpensive homemade quartz-tube atomizers which were developed for a study of tetraalkyllead compounds [3].

These atomizers consist of a quartz tube wrapped with heating wire or a slotted quartz tube held in an air—acetylene flame. The relative detection limits of the wire-wrapped quartz tube and the Perkin-Elmer HGA 2100 were the same, whereas that of the slotted quartz tube was 20 times greater in comparative tests with MMT. Thus, if desired, a quartz tube wrapped in heating wire can be used in place of the commercial furnace with no penalty to be paid in detection limit.

It is interesting to note that MMT atomizes readily giving free manganese atoms at the relatively low temperature of 1000°C attainable with the heating wire wrapped quartz tube atomizer. This temperature is in marked contrast to 2700°C often recommended as the atomization temperature in the commercial graphite furnace atomizers. In this regard, it was found necessary, for maximum sensitivity, to align the manganese hollow-cathode lamp so that the optical beam just grazed the inner surface of the quartz tube nearest the gas inlet. Sensitivities obtained in the center of the tube are greatly reduced.

Analysis of air samples

Air samples of up to 15 m^3 were taken at a variety of locations on the streets of Toronto. No MMT was detected in any sample (detection limit 0.05 ng m^{-3}). This compares with 14 ng m^{-3} for total tetraalkyllead compounds found in street air in similar locations. Samples were then collected in an underground car-park beneath the Chemistry and Physics buildings at the University of Toronto. In a few of these samples MMT was detected.

Figure 2 shows the chromatograms obtained when a calibration standard

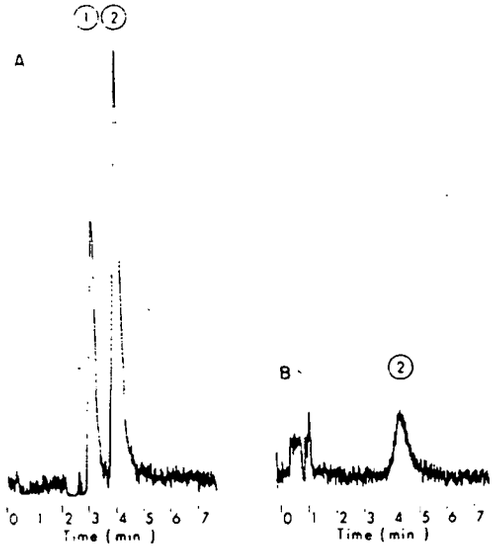

Fig. 2. (A) Chromatograph for standard sample containing MMT and CMT. (B) Chromatogram of an air sample taken in an underground car-park.

containing CMT which can be used as an internal reference if desired [2]) and MMT were run according to the proposed procedure. The level of MMT in the air sample was 0.3 ng m^{-3}. Other samples yielded values between 0.1 and 0.3 ng m^{-3}. It should be noted that the retention times of the MMT peak shown in the chromatogram for the air sample and the calibration standard are slightly different (ca. 0.2 min). It was not uncommon to have this variation which reflects the reproducibility problems found when sample traps are used.

It is interesting to speculate as to why the MMT levels in air are so much lower than those of tetraalkyllead when it is likely that a large fraction of the vehicles are using "unleaded" gasoline containing MMT. To this end, a cursory study of MMT in air was attempted.

MMT, liquid and vapor, was injected into clear 2-l glass bottles kept in the light and in the dark. Samples were then taken of the contained air and isooctane rinsings of the bottle walls at various intervals. Results of these studies suggest that MMT is quickly decomposed in air, more quickly in the light than in the dark. This conclusion must be treated as highly qualitative since a number of problems were encountered during this work.

Regarding this experiment, it was particularly difficult to keep the MMT suspended in the air. There is a great tendency for this compound to condense out on the sides of the bottles. Apart from making sampling difficult, this calls into question whether such a system is valid for determining the stability of MMT in air. Obviously, a full-scale separate study (well beyond the scope of this work) would be very important and should be undertaken.

The relative low volatility of MMT (i.e. its tendency to condense out on

surfaces) may also be a reason for the very low levels detected in air samples even in an underground car-park. With this in mind, the filter papers used for filtering particulate samples from the air in the MMT gas sampling train were rinsed and in some cases refluxed with isooctane. The rinsings were then injected into the gas chromatograph as a test for the presence of MMT, but in no case was MMT detected.

It is important to point out that tests done on exhaust samples taken from cars which were burning "unleaded" gasoline showed no MMT (detection limit 0.1 ng m^{-3}). This is consistent with a similar study of tetraalkyllead compounds [3]. Thus most of the MMT detected in the air samples is due to evaporation and spillage, etc.

This work was supported by a grant from the Ontario Ministry of the Environment, Research Grants Program and by Perkin-Elmer Corporation, Norwalk, Connecticut who supplied all the atomic absorption equipment. Thanks are also due to the Canada Center for Inland Waters, Burlington, Ontario, who lent the gas chromatograph.

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THE INFLUENCE OF Mn_3O_4 FROM MMT COMBUSTION ON GASOLINE VEHICLE EMISSIONS

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INTRODUCTION

In a trial of the influence of MMT fuel additive upon gasoline engine exhaust emissions, the Ethyl Corporation ran two matched fleets of vehicles, identically tuned and equipped with three-way catalyst systems. One fleet ran on fuel containing MMT additive at a concentration of 0.03125 g Mn per gallon of fuel, the other on an identical fuel without additive (termed "clear" fuel). The vehicles using MMT-additive showed statistically significant lower emissions of NO_x and CO, and slightly higher HC, than those running on clear fuel. The influence of MMT in reducing NO_x and CO emissions was greater over 0-75,000 miles than over 0-50,000 miles, whilst the effect on hydrocarbon emissions was marginally less over the greater distance.

This report seeks to explain the reasons for the observed differences in terms of the known catalytic properties of manganese oxides derived from MMT combustion.

SCIENTIFIC BACKGROUND

The combustion of MMT fuel additive in the gasoline engine leads to formation principally of the manganese oxide Mn_3O_4; traces of Mn_2O_3 have also been reported (1,2). Manganese oxides are well known for their catalytic properties, particularly in relation to their ability to oxidize carbon monoxide to carbon dioxide (3,4).

Past work in the laboratory (5) has shown that addition of MMT to iso-octane fuel in a pulse flame apparatus leads to reduced emissions of NO and CO, with increased hydrocarbons. The decrease in NO and CO emissions appeared to be associated with wall deposits of manganese oxides.

At the time of that work (1977/8), I formulated a series of experiments in conjunction with Professor Harry W. Edwards (Colorado State University). In these laboratory experiments, carried out at University of Lancaster (U.K.), the catalytic activity of Mn_3O_4 with respect to NO chemistry was investigated (6). We found that even in the presence of atmospheric oxygen, Mn_3O_4 catalyzed the decomposition of NO at temperatures as low as 215-243 degrees C (419-469 F). Concentrations of NO_2 were unaffected.

Nitric oxide, NO, is formed in the gasoline engine primarily by combination of atmospheric nitrogen, N_2 and oxygen, O_2 in a complex chain mechanism. The reaction is favored by high temperatures for two reasons. Firstly, the formation reaction is more rapid at elevated temperatures, and secondly the equilibrium concentration of NO is highly temperature sensitive with highest concentrations formed at high temperatures. The equilibrium constant, K, for the reaction is given by equation (1), from ref. (7):

$$N_2 + O_2 \; < == > \; 2NO$$

$$K = \frac{X_{NO}}{X_{N_2}^{1/2} \; X_{O_2}^{1/2}} = 4.69 \exp(-21{,}600/RT) \qquad (1)$$

Where X represents the partial pressure. At temperatures of 1000 K (1341 F) and below, the equilibrium concentrations of NO are very low. However, much higher concentrations can exist as the decomposition reaction is very slow at these temperatures and the NO is essentially "frozen in". A catalyst which speeds the decomposition reaction will lead to low-temperature breakdown of NO. It appears that Mn_3O_4 is such a catalyst (6). If 482 C (900 F) is taken as a typical exhaust gas temperature, the equilibrium partial pressure of NO may be calculated from equation (1). At an oxygen concentration of 1%, this is less than 1 ppm. Thus, in the presence of a suitable catalyst, NO decomposition would be expected.

INFLUENCE OF Mn_3O_4 IN THE VEHICLE EXHAUST

Scenario for NO Decomposition in Vehicle Exhaust

As a simplification, I have assumed that in a vehicle travelling at 30 mph, fuel of composition C_8H_{14} is combusted at a rate of 21 miles per U.S. gallon. MMT additive is used at a concentration of 0.03125 g Mn per U.S. gallon. The exhaust gases pass through a 2 meter length of 3 inch internal diameter pipe, at a mean temperature of 482 C (900 F). As it is known that only a small proportion of Mn burned in the engine is emitted in the exhaust, it is assumed that the walls of this pipe become coated with Mn_3O_4.

Calculation of Influence Upon NO Concentrations

In my paper on NO decomposition over Mn_3O_4, the reaction was found to be first order in NO with Arrhenius parameters of E_{app} = 12.4 ± 0.2 kcal mol^{-1} and lnA(s) = 9.29 ± 0.24. Using the relationship,

$$k = A \exp(-E_{app}/RT) \qquad (2)$$

(where R is the gas constant and T the absolute temperature), the rate coefficient k at 482 C (755 K) may be calculated to have a value of 2.93 s^{-1}.

In the plug flow reactor used in our study, the integrated rate expression was as follows:

$$-k\ V/u = \ln (Ce/Ci) \qquad (3)$$

in which V = reactor volume
u = volume flow rate
Ce = steady state exit concentration
Ci = steady state inlet concentration

In the vehicle exhaust scenario, the ratio V/u, the residence time, is calculated to be 0.24s.

Then, calculation gives

$$\frac{Ce}{Ci} = 0.50 \quad \text{at 900 F}$$

This result implies 50% decomposition of NO and relates to conditions in our reactor. The precise surface area of Mn_3O_4 was not determined in our studies, but a minimum value was estimated as 0.005 $m^2\ g^{-1}$. As the charge was 4.03 g of Mn_3O_4, the total surface area (minimum estimate) is 202 cm^2. In the vehicle exhaust scenario, the area of exhaust pipe surface is 4,800 cm^2. The actual area of Mn_3O_4 deposits coating this surface will be appreciably greater as the surface deposit will have an uneven microstructure, and may be 10 fold, or more times this value. It is clear, therefore, that even if the specific surface area of Mn_3O_4 in our experiments is appreciably greater than the reported minimum value, the exposed surface area in the auto exhaust system is of a similar magnitude, assuming an even coverage of manganese oxide. Further decomposition of NO may also occur as a result of contact with Mn_3O_4 deposits in the three-way catalyst.

It is also possible to calculate the surface area of Mn_3O_4 aerosol in the exhaust gases. If spherical particles of diameter 10nm (typical of primary exhaust aerosol) are assumed, the surface area of aerosol present at any instant in the 2 meter length of exhaust is ca. 5cm^2, and thus much lower than that of the coated surfaces.

Surface-catalyzed reactions, at low conversions, typically show a mass conversion rate linearly proportional to the active surface area of the catalyst (3). At higher conversions in a plug flow reactor, a less than linear dependence would be expected, due to a reduction in

NO partial pressure as the gases pass through the reactor. The results of the calculation therefore lead me to the view that a coating of Mn_3O_4 in the vehicle exhaust could lead to a substantial reduction of the NO concentration discharged from the engine cylinder. This process will be accompanied by formation of O_2.

$$2NO \rightarrow N_2 + O_2$$

Reductions of NO of the magnitude suggested above are quantitatively consistent with the observations of Otto and Sulak (5), who found a reduction in NO of 91% after 800 hours in experiments with the pulse flame apparatus with a constant level of CO.

Vehicle trials carried out by Ethyl Corporation have shown an increasing difference in emissions of both NO and CO with mileage between vehicles using clear fuel and fuel with MMT. This trend of improved performance in the MMT-fuel vehicles is consistent with the build-up of a surface coverage of Mn_3O_4 in the exhaust system.

Influence on Concentrations of CO

Our experiments did not include the influences of CO and HC. There is, however, substantial literature demonstrating the catalytic influence of manganese oxides, including Mn_3O_4 on oxidation of CO (e.g. ref.: 8)

$$2CO + O_2 \rightarrow 2CO_2$$

Indeed, it is suggested (9) that Mn_3O_4 is the phase most active in this process. There are also precedents for the catalysis of CO oxidation by NO on metal oxides, including those of manganese (e.g. ref.: 10).

$$2NO + 2CO \rightarrow N_2 + 2CO_2$$

Whilst it is not possible to predict at this stage by which mechanism the effect occurs, it seems very probable that Mn_3O_4 is catalyzing CO oxidation and effecting an improvement in exhaust composition as a result. This effect was seen also in the work of Otto and Sulak (5) in the pulse flame apparatus. There are, to my knowledge, no detailed kinetic studies of these reactions available in the literature from which quantitative predictions might be made.

CONCLUSIONS

Operation of vehicles on fuel containing MMT additive causes deposition of a surface coating of the manganese oxide, Mn_3O_4 on the internal surface of the exhaust system. This coating has catalytic properties which cause decomposition of nitric oxide to N_2 and O_2, and may also catalyze loss of carbon monoxide by reaction with O_2, or with NO. This catalytic effect leads to an improvement in exhaust gas quality relative to vehicles running on clear fuel which increases with the age of the vehicle as the catalytic coverage of Mn_3O_4 grows.

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APPENDIX 10

AN ASSESSMENT OF THE EFFECT OF MMT ON LIGHT-DUTY VEHICLE EXHAUST EMISSIONS IN THE CANADIAN ENVIRONMENT

Prepared by a Working Group
Under the Jurisdiction of
The CGSB Gasoline and
Alternative Automotive
Fuels Committee

April 4, 1986

C O N T E N T S

SUMMARY

A working group of the CGSB Gasoline and Alternative Automotive Fuels Committee has reviewed the literature on the effect of manganese antiknock (MMT) on tailpipe emissions from light duty vehicles. It has concluded that the presence of MMT in commercial gasolines has no detectable effect on carbon monoxide or oxides of nitrogen emissions. Hydrocarbon emissions should increase on average from 0.03 to 0.11 g/mile due to the presence of MMT.

The emission-control systems developed in the U.S. and elsewhere to meet the present U.S. limit of 0.41 g/mile appear unlikely to suffer damage from exposure to MMT treated fuels. These emission-control systems will almost certainly be utilized on Canadian vehicles when this standard is adopted in Canada in 1988. A deterioration in HC control may be detectable over an 80,000 km (50,000 mile) test, but this would have no significant effect on overall average air quality. The major improvement (79.5%) will be due to the tightening of HC limits from 2.0 g/mile to 0.41 g/mile. Assuming the 0.11 g/mile increase, the continued presence of MMT in commercial gasolines would still permit a 74.0% improvement over the present standard.

On this basis, the working group recommends the retention of MMT at current levels in Canadian unleaded gasoline. Should changes in emission-control systems be introduced in the future, the question of their response to MMT should again be addressed.

* * * * *

INTRODUCTION

On August 3, 1985, the Department of Transport published, in the Canada Gazette Part 1, a notice proposing new emission standards for light-duty vehicles and light-duty trucks. The proposal would reduce the present allowable level of unburned hydrocarbon (HC) tailpipe emissions from 2.0 g/mile to 0.41 g/mile for light-duty vehicles and 0.8 g/mile for light-duty trucks, to take effect September 1, 1987, with the introduction of the 1988 model year. Vehicles will be tested for compliance with these standards using EPA procedures with accumulated mileage utilizing MMT-free gasoline.

In adopting these tighter standards, the use of the octane enhancer methylcyclopentadienyl manganese tricarbonyl (MMT*) in commercial pump gasoline would be continued at the current Canadian General Standards Board (CGSB) limit of 18 mg Mn/litre unless its continued use could be shown to have unacceptable adverse effects on health or on the environment.

There are sound economic reasons for the retention of MMT in Canadian unleaded gasolines. This useful antiknock agent saves energy and money in meeting the octane requirements of the Canadian automobile population. However, the effects of MMT on the Canadian environment, with particular reference to the new standard, have not been fully evaluated.

To assess the potential effects of manganese compounds on vehicle emissions and on emission system durability in Canada, Environment Canada requested assistance from the Canadian General Standards Board Petroleum Committee. The CGSB Gasoline and Alternative Fuels Committee subsequently appointed a working group to carry out this activity. The working group comprised members from the petroleum industry, the vehicle manufacturers and the federal government (Appendix I). Comments and advice were solicited from other interested parties such as health and consumer groups (Appendix II).

OBJECTIVES

The objectives of the working group, outlined in a letter (dated 27/5/85) by the Assistant Deputy Minister, Environmental Protection Service, Dr. R.W. Slater, to Dr. R. Whyte, Chairman of the Petroleum Committee, were as follows:

i) to review and report on the current state of knowledge respecting MMT effects on vehicle emissions,

*Ethyl Corporation's TM

ii) to forecast the possible impact on the vehicle emission control systems likely to be in use in the period 1988-2000,

iii) to assist Environment Canada in documenting the impact that continued use of MMT may have on air quality.

This report presents the findings and judgments of the working group.

METHOD

To accomplish this task, the working group gathered the information through the following methods.

(i) Reports

Relevant reports on MMT were compiled and reviewed (see bibliography). It was noted that very little work had been reported since 1978 when the use of MMT in unleaded gasoline was disallowed by the U.S. Environmental Protection Agency (EPA).

(ii) Test Programs

The results of two test programs were reviewed by the working group; the "CRC MMT Field Test Program" reported in June 1979; and a recent in-use vehicle surveillance test program conducted by Environment Canada which tested 1983 to 1985 model year Cadillacs, calibrated to U.S. emission specifications (References 1 and 2).

(iii) Vehicle Manufacturers

Information was requested from both the Motor Vehicle Manufacturers Association (MVMA) and the Automobile Importers of Canada (AIC) member companies (Appendix III and IV), as to how current emission-control systems were behaving in Canada and how future emission-control systems would behave if MMT were retained in gasoline at the current CGSB limits.

MMT EFFECTS ON VEHICLE EMISSIONS

The working group realized from the outset that very few studies had been reported on MMT and its effects on exhaust emissions, since it was disallowed by the EPA in the U.S. for use in unleaded gasoline in 1978. Subsequent applications for a waiver by Ethyl Corporation to use MMT in U.S. unleaded gasolines were not granted (Appendix V).

An extensive test program was carried out by the Co-ordinating Research Council (CRC) in 1977-78, and more recently Environment Canada conducted an in-house study, both of which have been extensively reviewed by the working group.

1) CRC MMT FIELD TEST PROGRAM

The CRC program involved 63 vehicles. The objective of the CRC program was to study the effects of two concentrations of manganese on exhaust emissions and, to compare these results with those for clear fuel. The 63 vehicles were divided into three matched fleets. The first fleet was operated on clear gasoline, the second oh gasoline which contained MMT at a level of 1/32 gMn/USG (8.25 mgMn/L), and the third, on gasoline which contained MMT at 1/16 gMn/USG (16.5 mgMn/L). Within each fleet, seven car models were tested in triplicate. In other words, nine replicate cars of each of the seven car models were tested resulting in a 63-car test program.

Of the seven car models studied in the CRC program, only two featured three-way catalysts (TWC) and closed-loop fuel systems, the types likely to be in use in Canada by the 1988 model year. These two models were a Pontiac Sunbird and a Volvo 242, the former being electronic feedback carbureted, and the latter having feedback electronic fuel injection. Five car models featured conventional oxidation catalysts (COC) and open-loop fuel systems. All of these vehicles were essentially production models which had been calibrated to meet 1977-78 California emission standards of 0.41 g/mile HC, 9.0 g/mile carbon monoxide (CO) and 1.5 g/mile oxides of nitrogen (NOx). These vehicles had been selected by their respective manufacturer since they were considered to be sensitive to MMT and/or they were representative of future high-volume powertrains.

The presence of MMT was found on average to be statistically significant only for HC, and did not affect CO or NOx emissions. While the vehicles exhibited varying initial HC emissions, all were calibrated to meet a 0.41 g/mile hydrocarbon standard.

The study found that, on average for all vehicles, the tailpipe HC emissions increased by 0.11 g/mile relative to clear fuel, after the 50,000-mile test period while using MMT at 1/16 gMn/USG (16.5 mg Mn/litre) in the fuel.

The data from the two TWC model cars were not consistent, and ranged from zero to 0.17 g/mile difference in HC emissions relative to clear gasoline at 50,000 miles. The Volvo cars, produced the lowest HC emissions and had the smallest response to MMT. The average HC increase relative to clear gasoline was 0.03 g/mile when using 1/16 g Mn/USG (Appendix VI - Composite Results Prepared by the Working Group). The applicability of this result to all vehicles equipped with fuel injection and TWC is questionable since TWC electronic fuel-injection control system may not be more efficient than a COC system at controlling the increased HC emissions due to MMT which are generated in the combustion chamber. Electronic fuel injection will allow better control of the air-fuel ratio, and this will only reduce the 'spread' of HC emissions when compared to carbureted vehicles. However, of the cars tested in the study, the Volvo fuel and emission-control system was closest to that likely to be sold in Canada during the 1988 model year and beyond.

In addition, there was no indication of catalytic converter plugging with any of the fuels in the test program, as monitored by the pressure drop across the catalyst. In no instances was the fuel economy of the vehicles of the CRC test fleet affected by the MMT in the gasoline.

The CRC study also found that the use of MMT resulted in a catalytic converter efficiency increase of 2 to 3%, although this was not enough to offset increases in engine-out emissions.

2) ENVIRONMENT CANADA STUDY

In 1984 Environment Canada, in order to generate HC emission data, conducted surveillance tests of in-use vehicles calibrated to U.S. emission control specifications which had been operating on Canadian unleaded gasoline containing MMT at levels of up to 18 mg Mn/L. The cars, model years 1983, '84 and'85, featured electronic throttle-body fuel-injection, an oxidation catalyst working in conjunction with a three-way catalyst, and closed-loop fuel control. Tests were not undertaken on misfueled vehicles or vehicles with malfunctioning fuel-control systems. Fifteen rented in-use Cadillac cars equipped with 4.1-litre engines were tested and sixteen acceptable Federal Test Procedure (FTP) tests were performed.

The results indicated that, when using data generated by a different vehicle at each test point, these cars would not exceed the proposed hydrocarbon emission level of 0.41 g/mile when test results were extrapolated to 50,000 miles. However, any conclusion regarding HC emission increase, if any, due to MMT in the gasoline, could not be established due to the absence of base-line data for similar vehicles operated on clear fuels.

The major conclusions based on these two test programs may be summarized as follows:

1. Based on all cars tested in the CRC study, the average increase in tailpipe HC emissions at 50,000 miles using MMT at 1/16 g Mn/USG was 0.11 g/mile higher than for clear fuel. This may be taken as the best average case.

2. Based only on the two TWC car tests in the CRC study, the average HC increase for the 1/16 g Mn/USG fuel ranged from zero to 0.17 g/mile higher than for clear fuel, at 50,000 miles.

3. For the Volvo system, which was assumed to be closest to the current and future technology, the average HC increase relative to clear fuel was 0.03 g/mile (Working group analysis of Volvo data). This may be taken as the best case.

4. All cars in the Environment Canada study met the proposed HC standard of 0.41 g/mile on fuel containing MMT, when extrapolated to 50,000 miles. The effect of MMT on vehicle HC emissions was inconclusive since no clear fuel base line data were obtained. However, there were no indications that the MMT effect is greater than that indicated in the CRC report.

5. The effect of MMT on HC emissions may be of the same order of magnitude as the test uncertainty in a small fleet test. Therefore, it may not be possible to measure statistically significant differences due to MMT, especially in cases where the majority of cars meet the HC standard.

IMPACT ON FUTURE EMISSION-CONTROL SYSTEMS

VEHICLE MANUFACTURERS

The MVMA and AIC have been asked to predict how future emission-control systems would behave if MMT were retained in unleaded gasoline. Based upon limited data available, there is no evidence to suggest that emission-control systems that have been developed for today's market would experience adverse effects with regard to function or performance from the use of MMT in gasoline. Current systems do not indicate that durability is lower in Canada, where MMT is used, than in the United States where MMT has been disallowed in unleaded gasoline. Members of MVMA and AIC indicate that manufacturers' Canadian warranty claims on emission components are comparable to the U.S.

It must be noted however that the effect of MMT on emission-control system components may be subtle and may never result in a failure which would be apparent through analysis of warranty claims. Degradation of the emission performance of a vehicle does not necessarily manifest itself in the form of poor driveability or performance yet; when emission tested, the vehicle may display an increase in emission levels above those to which the vehicle was designed.

The durability of critical emission-control components related to MMT use may change in the future. This may be reflected in the manufacturers' warranty records. Therefore, short of complete emission measurements, periodic reviews of warranty records may be necessary to determine if future action on MMT is required.

In the past, some researchers noted that MMT tended to form deposits, typically manganese oxide, which coated the oxygen sensor in the exhaust system of closed-loop type cars. Depending upon the design of the oxygen sensor, deposition of manganese oxide could impede the sensor's ability to function properly in controlling exhaust emissions, as was evidenced in the CRC program already cited. A failure of this nature may or may not be accompanied by a gross deterioration in vehicle driveability.

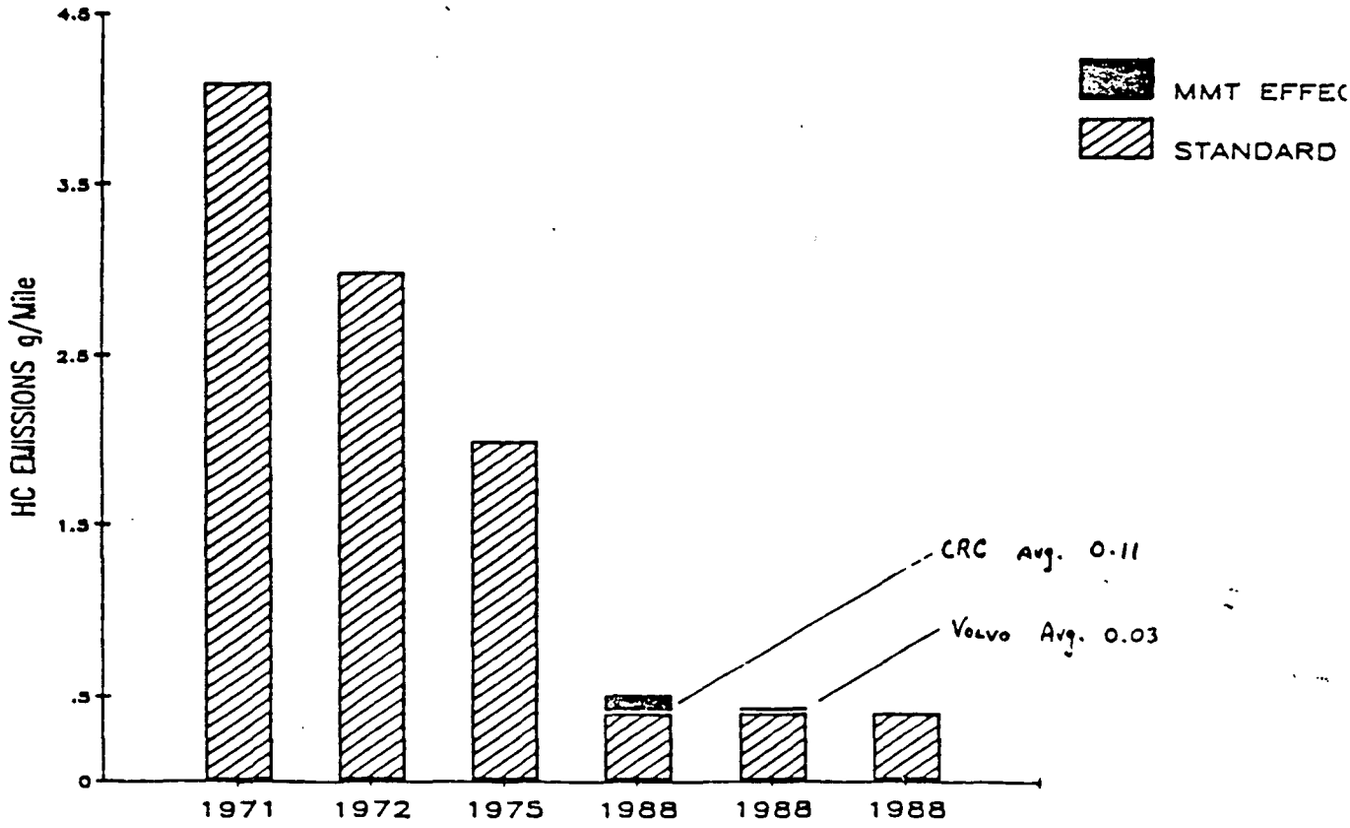
The working group has considered that further test programs on vehicles using today's equipment could well provide additional information. However, this working group has received no evidence to suggest that an average increase would be greater than 0.11 g/mile. Future technologies suggest that exhaust emission-control systems will be more effective at controlling emissions sensitive to the effects of MMT,and therefore further large-scale testing may be of limited value.

The working group would recommend a test program only in the event that future emission-control technology was demonstrated to be adversely affected by MMT. The CGSB Committee on Gasoline and Alternative Automotive Fuels would be receptive to any new data generated on MMT fuels.

GENERAL DISCUSSION

The effect of MMT usage on HC emissions is shown in the graph below. The HC emission reductions since 1971 have also been indicated.

Canadian Hydrocarbon Emission Standards



COMMENTS

1) The proposed standard of 0.41 g/mile represents a reduction of 79.5% from the present standard of 2.0 g/mile.

2) Assuming that MMT increases HC emissions on average by 0.11 g/mile relative to clear fuel, an actual HC level of 0.52 g/mile (0.41 + 0.11) represents an average reduction of 74.0% from the present standard.

3) Assuming that MMT increases HC emissions by 0.03 g/mile relative to clear fuel, an actual HC level of 0.44 g/mile (0.41 + 0.03) represents an average reduction of 78.0% from the present standard.

The actual average increase in HC tailpipe emissions should lie somewhere between these two values and consequently the working group cannot see any significant benefit in removing MMT from unleaded gasoline in Canada based on tailpipe HC emission levels.

IMPACT ON AIR QUALITY

The working group has not attempted to carry out any very rigorous study on the effect of retaining MMT on overall air quality. Environment Canada have, however, published a "National Inventory of Natural Sources and Emissions of Organic Compounds" in which the total man-made emissions of hydrocarbons are estimated as 8.4% of the whole (Reference 3 and 4). Of these, 16% are attributed to light-duty vehicles.

It can thus be calculated that reducing the hydrocarbon standard from 2.0 g/mile to 0.41 g/mile (79.5%) will result in a reduction in HC burden of 79.5 x 0.16 x 0.084 = 1.07%.

In the worst case, an increase of 0.11 g/mile due to MMT, the overall reduction would be 74%. This equates to an improvement in air quality of 0.99%. In the best case, an increase of 0.03 g/mile due to MMT, the emission reduction would be 78%, equivalent to an improvement in air quality of 1.05%.

NATIONAL ENVIRONMENTAL INVENTORY SYSTEM (NEIS)

The level of HC tailpipe emission increase due to MMT will be required by the Department of the Environment in order to calculate an emission factor for use in NEIS. Analysis of the data has indicated that in future, with the majority of new cars being fitted with three-way catalysts, fuel injection and closed-loop fuel control systems,the average increase in tailpipe HC emissions could be as high as 0.11 g/mile for post-1988 model cars. It is recommended that this value be used by the Department of the Environment to calculate the emission factor.

CONCLUSIONS

The major conclusions of the working group based on the analysis of currently available data are as follows:

1) When using MMT the average increase in tailpipe HC emissions over clear fuel should range from 0.03 to 0.11 g/mile for the 1988 model year light-duty vehicles.

2) The proposed standard would reduce the allowable tailpipe HC emissions from 2.0 g/mile to 0.41 g/mile. With clear fuel this is a HC reduction of 79.5%. With MMT the average reduction would be from 74% to 78%.

3) The use of MMT at current CGSB levels does not significantly compromise emission-control system operation or component durability.

4) The working group has not attempted to carry out any rigorous study on the effect of retaining MMT on overall air quality. However, since it is estimated that vehicle emissions contribute only 1.35% of hydrocarbons present in the atmosphere, the effects of MMT is considered miniscule.

RECOMMENDATIONS

1) Based on the findings in this report, the members of the working group recommend that MMT be retained at current levels as an octane enhancer in unleaded gasoline.

2) The members of the working group recommend that the issue be re-examined in the event that future emission-control technology or the generation of additional data, should show adverse effect due to the use of MMT beyond those recognized in this report.

Signed... K.A. Heller .....

Chairman of Working Group

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APPENDIX I

CGSB WORKING GROUP ON MMT IN GASOLINE

NAME	AFFILIATION
K. Miller	Petro-Canada Products Inc.
R. Solman	Environment Canada
P. Sarvos	Shell Canada Ltd.
G. Caldwell	Motor Vehicle Manufacturers' Association
R. Falkiner	Esso Petroleum Canada
J. Collins	Ethyl Canada Inc.
J. Dowell	Energy, Mines and Resources
M. Mokry	Automobile Importers of Canada

The Canadian Association
for Children and Adults
with Learning Disabilities

L'Association canadienne
pour enfants et adultes ayant
des troubles d'apprentissage

Maison Kildare House, 323 Chapel, Suite 101, 102, Ottawa, Ontario K1N 7Z2 (613) 238-5721

August 27, 1985

Mr. J.O. Cliffe, Chairman
C.G.S.B. Committee on Gasoline & AAF
432 Martin Grove Road
Islington, Ontario
M9B 4M2

Dear Mr. Cliffe:

Thank you for your letter and attached information concerning the working group under the Canadian General Standards Board investigating the long-term effects of the continued use of MMT in unleaded gasoline.

I'm sorry I was unable to contact you or attend, but I was out of the province in July on vacation. As you know the conclusion stated in the report IP-8 The Technology and Costs of Control Automobile Emissions in Canada is as follows: "The consensus of EPA and the automotive industry is that MMT produces significant adverse effects on HC emissions." The authors were especially concerned about failure of sensors and other effects on the catalyst.

Our primary concern in the long-term use of MMT is with health, particularly its neurotoxic attributes in the case of young and unborn children. I have communicated these concerns directly to Mr. Hazra of EPS, and Dr. Joseph Ruddick of the Health Protection Branch of Health and Welfare.

Sincerely yours,

Barbara McElgunn

Barbara McElgunn
Research and Liaison Officer (Health)

432 Martin Grove Road,
Islington, Ontario
M9B 4M2

September 3, 1985

Mrs. Barbara McElgunn,
The Canadian Association for
Children & Adults with
Learning Disabilities,
Kildare House,
323 Chapel, Suite 101, 102
Ottawa, Ontario
K1N 7Z2

Dear Mrs. McElgunn:

Thank you for your letter of August 27th concerning the Working Group of C.G.S.B. set up under the auspices of my committee to review the data available on MMT in gasoline and its effects on automobile emissions. I have passed it on to Mr. Keith Miller of PetroCanada who is the chairman of the Working Group.

I was, of course, aware of your earlier contact with Mr. Hazra since it was he who had given me your address.

I understand that the Working Group has held one meeting since the inaugural one and will be meeting again sometime in October. By copy of this letter, I am asking Mr. Miller to ensure that you remain up to date on any meeting notes which may have been issued. If you need to contact him, his phone number is 416-445-9113. I will, myself, be out of town most of October and the first two weeks of November.

Yours very truly,

Jack Cliffe

J.O. Cliffe

cc. Mr. K. Miller,
PetroCanada Products Inc.,
240 Duncan Mill Road,
Don Mills, Ontario
M3B 3B2